Journal of Forensic Medicine

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The Place of Forensic Medicine

In the Post-Graduate Medical Curriculum

The Second World Conference on Medical Education will be held under the auspices of the World Medical Association in Chicago at the end of August next year. Collaborating organizations include, *inter alia,* the International Association of Universities.

The teaching of forensic medicine is the subject of constant debate. In 1955 we published an editorial and a symposium devoted to the undergraduate and post-graduate problems involved.* Since then, much advance and experiment has proceeded in academic circles and it would be a great pity if this important subject were, by an oversight, excluded from consideration and discussion at the Chicago Congress next year.

Teachers and practitioners of forensic medicine will agree that the undergraduate curriculum cannot be expected to produce a medico-legal expert. The primary aim of the medical curriculum is to produce competent clinical practitioners, and this must not be lost sight of by the experts in the various fields of specialization. On the other hand, medical practitioners are constantly and increasingly involved in medico-legal problems from the moment of their graduation. It therefore becomes necessary to ensure that, as part of the equipment with which the fledgling doctor embarks on his career, he has a firm grasp of fundamental principles and that adequate provision is made for his continuing post-graduate training—apart from the extra-mural activities of professional societies concerned with these matters.

Naturally, different countries will have different national problems, but useful suggestions may grow out of an exchange of experience in this field.

* This *Journal,* 1955, Vol. 2, No. 3, 129, 132.

It would be very desirable, at an international level of discussion such as will be provided next year, to explore the possibility of reaching some agreement on the fundamentally necessary content of the forensic curriculum. Such a debate would clarify many issues and provide much needed guidance for those concerned with post-graduate forensic training.

A discussion on a post-graduate career in medicine would be seriously incomplete without a considered debate on the potentialities of forensic medicine. It is therefore to be hoped that there will be an adequate expression of opinion from teachers of Forensic Medicine at the Chicago meeting next August.

Criminal Thallium Poisoning

R. Matthys* and F. Thomas†

From the Department of Legal Medicine and the Neuro-Psychiatric Clinic of the University of Ghent, Ghent, Belgium

A survey of the French and German literature brings out a sharp contrast between the very few cases of thallium poisoning reported in the French literature (these cases being rare in French-speaking countries) and the abundance of cases reported in the German literature (these cases being frequent in Germany). This contrast is probably due to the fact that the Germans were the first to put on the market (about 1920) the thallium rodenticides, which were produced at a low price from industrial by-products. It is therefore not surprising that the Germans should have been the first to use thallium criminally.

Why did the new rodenticide so quickly replace those already on the market and of proven efficiency? Schrader and Knorr provide us with the explanation. In rural districts the dead rats are often eaten by the pigs. The latter offer little resistance to phosphorus intoxication and may eventually succumb when they devour cadavers of rats poisoned by phosphorus. On the contrary, with the new rodenticide, no accidents occur, not because of a relative immunity of pigs to thallium, but rather because, at the time the rat dies, most of the poison, thanks to its high solubility, has already been eliminated.

The first case of criminal poisoning by thallium was reported in 1928 separately by Haberda and by Kaps. The diagnosis was not established during life. The symptoms had been attributed to arsenic poisoning, a mistake which has frequently occurred since. During the last 25 years, cases of thallium poisoning have increased alarmingly in number. Entirely unknown in Belgium before the last war, they are now frequent. We have seen 20 cases since 1947, 16 of them in Flanders. We will report only one of them as illustrative of the whole group.

The victim (Figs. 1 and 2) was a man of 40 years, a fitter by profession, married and the father of 5 daughters. One day, towards the end of 1947, he noticed the bad taste of the coffee he had taken with him to his work. He vomited and complained of diarrhoea and colicky pains. The following day he was better.

The symptoms which finally necessitated hospitalization began in mid-January 1948. One day, after eating sandwiches containing liver paste, he vomited and

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suffered from severe colic. The next day, he complained of pain in the lumbar region and in the legs. The abdominal pains persisted. He had a feeling of 'needles in the stomach.' He vomited after each meal. On the fourth day he was obliged to stay in bed. He experienced violent paresthesias in the toes, first on the left side, then also on the right. Lesser paresthesias occurred in the hands. The pain was so acute that the family doctor resorted to morphine. The patient was very constipated and had no appetite. He further suffered from total insomnia.

At the beginning of February 1948, massive loss of hair occurred, including the hair on the legs.

Neurological Examination. We will confine ourselves to the positive findings. The muscular strength of both arms and legs was greatly decreased at all levels. The muscles showed hypotonia, without atrophy. Sensation was also disturbed, viz. tactile hypoesthesia, more marked on the right than the left leg. Palpation of the skeletal muscles was extremely painful. The patellar and ankle jerks were brisk and symmetrically equal. Lumbar puncture revealed no spinal fluid changes whatsoever. Chronaxie was increased in both legs, mainly distally.

Slow recovery set in, the treatment being limited to vitamin B administration. The patient resumed his work in August.

We were, for the first time, called upon to examine him in April 1950. He had left his wife 2 years ago and was finally compelled to bring charges against her because she had sued him for an alimony, notwithstanding having herself admitted poisoning him! Except for a few positive neurological signs, the patient may be described as having completely recovered. The hair has re-grown. All laboratory findings were normal. No toxicological examination was suggested by us, since it was obviously too late to carry this out.

To sum up, we were confronted with a sensory-motor polyneuritis preceded by gastro-intestinal disturbances and accompanied by a pathognomonic symptom—the alopecia. The latter, occurring in a patient suffering from unbearable pain in the big toe, led us to the diagnosis.

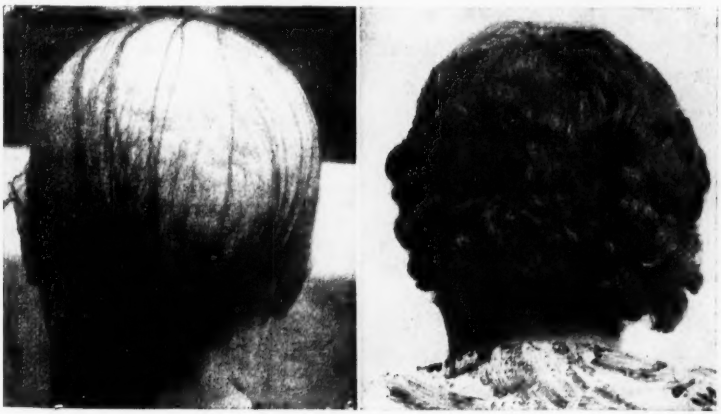
During the preliminary inquiry the patient's wife unhesitatingly confessed that she had put compost in her husband's coffee in August 1947, and that, in December 1947, she had bought a tube of Zelio paste, the contents of which she had spread on her husband's sandwiches. She had done this twice, using in all 2 tubes. The Court of Assizes sentenced her to 4 years' imprisonment.

Figs. 1. *and* 2. Thallium poisoning. A man 40 years of age. *Left:* The alopecia is complete.
Right: Condition after recovery.

Figs. 3–10 illustrate the condition of the survivors of a case of quadruple criminal thallium poisoning, with 2 deaths. It can be summarized as follows:

A woman, aged 52, accompanied by her son, aged 17, came to Ghent to consult us. They were losing their hair by the handful. Furthermore, they complained of 'rheumatism.' 'A curse has struck our family,' the woman said. 'My husband, aged 44, died only 2 weeks ago; his brother, who was 48, died nearly at the same time.'

Figs. 3 *and* 4. Thallium poisoning. A woman 52 years of age.
Left: The alopecia is nearly complete.
Right: Condition after recovery.



Figs. 5 *and* 6. The back of the head of the patient in Figs. 3 and 4.

The anamnesis made it possible in each case to constitute the typical clinical entity of thallium poisoning. Exhumation of the 2 deceased relatives, suggested by us, was ordered without delay. As the latter had survived only a relatively short time, the chemical investigations were positive. Thallium was readily found in the gastro-intestinal tract of both bodies, as also in the liver, etc. We wish to emphasize that the 2 deceased persons had presented a symptomatology which, though classical, had (as is often the case) completely led astray the family doctor.

As mentioned before, the first case of criminal thallium poisoning was described by Kaps, a clinician, in 1927, and one year later by Haberda, who had performed the post-mortem examination. The crime had not been detected and would undoubtedly have remained unknown if it had not been that the widow of the victim, who felt pangs of remorse, confessed it. In the light of our present knowledge, the clinical symptoms were most characteristic.

The Clinical Picture of Thallium Poisoning

The first signs, whether the case is acute or sub-acute, are either gastro-intestinal or neurological. Within a few hours of the ingestion, nausea and vomiting set in, accompanied by violent colics. Sometimes there is diarrhoea. In all cases extreme constipation follows and quickly dominates the abdominal symptomatology. In some cases it even leads to a diagnosis of intestinal occlusion. When a small dose of poison is administered repeatedly, the gastro-intestinal disturbances may pass nearly unnoticed.

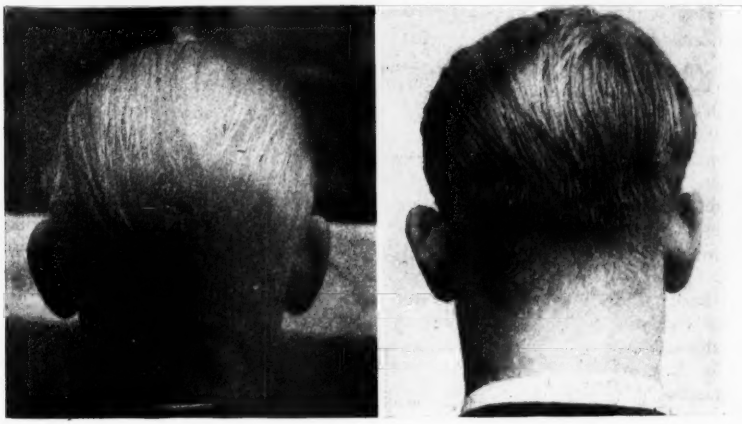
Figs. 7 *and* 8. Thallium poisoning. A boy of 17 years (son of preceding patient).
Left: The alopecia is nearly complete.
Right: Condition after recovery.

The first complaint, within a few hours to a couple of days after the ingestion of the poisoned food, is one of violent pain in the extremities of the limbs and especially in the *big toe*. They are the expression of the early damage to the peripheral nervous system in the form of polyneuritis, such as can also be caused by other toxic substances, viz. alcohol, arsenic, sulphonamides, etc.

The sensory disturbances which, in the first stages, remain confined to a vague, disagreeable feeling, often assume the form of painful paresthesias which are exacerbated progressively until they become unbearable and deprive the patient of sleep and rest both by day and by night. The complaints are variously described by the sufferers: frozen limbs, frozen feet, feeling as though the legs were enveloped in wet felt, or laid on glass splinters, or as if the toes were being cut off.

The topographic distribution of the sensory phenomena is almost the same in all patients. They start in the sole of the feet and ascend towards the trunk. Before the latter is reached the same process repeats itself in the hand, though to a lesser degree. A remarkable feature already described is that, in many cases, the whole drama may first manifest itself in the form of violent pain in the big toe, just as in gout. In some cases the clinical picture, initially monosymptomatic, led to the administration of an electro-convulsive treatment, so much did the complaints suggest some non-organic, mental origin.

These painful paresthesias are spontaneous and are usually exacerbated during the night. Sometimes, though, they must be provoked to manifest themselves. Palpation of the muscles, pressure along the peripheral nerves, even the weight of the bed clothes, can give rise to unbearable pain.



Figs. 9 *and* 10. The back of the head of the patient in Figs. 7 and 8.

Motor disturbances appear next. They express themselves in the form of tiredness in the legs. Walking then becomes more difficult as the muscular weakness develops into a paresis, or even a complete paralysis of the lower limbs. The same may occur to a lesser extent to the upper limbs.

The neurologic picture is therefore that of a sensory-motor polyneuritis of the flaccid ascending type, but predominantly painful. The neurological examination further reveals disturbance of deep and combined sensation, because the poison has not only damaged the peripheral nerve fibres but also the tracts of Goll and Burdach. The deep reflexes are sometimes increased in the first stages, only to disappear later on. This areflexia is due to the interruption of the reflex pathway, in its centripetal as well as in its centrifugal portion.

Among the cardinal symptoms, the most striking is undoubtedly the *loss of hair.* It generally occurs between the 14th and the 18th day, sometimes a little earlier. Prick has noticed it as early as the 10th day. It is massive, the hair falling by the handful on the slightest traction. In a few days (in the course of the 3rd week) the patient becomes completely bald. Not only does the hair of the scalp fall out, but the eyebrows are also involved in the process. The French clinicians speak of *le signe du sourcil.* Only the external two-thirds are affected. The inner third, which is phylogenetically much older, remains almost intact. Pubic and axillary hair, sometimes but not always, are also lost. The dermatologists tell us that females who wish to get rid of axillary or facial hair and, for this purpose, resort to thallium compounds, lose their hair instead, the undesirable hypertrichosis remaining unaffected.

About a month after it has been lost, the hair begins again to grow. After a few weeks it is as abundant as before. Sometimes a slight change occurs either in colour or in texture.

In a recent and convincingly illustrated paper (*A Simple Method of Early Diagnosis of Thallium Poisoning Based on Changes in the Hair Roots,* Bulletin de la Société des Amis des Sciences et des Lettres de Poznan, série C, livraison VII, 1957), as also in 2 excellent preliminary notes in Polish with English summaries, W. Widy describes remarkable changes in the hair roots which had escaped notice until now. They are, he says, easily detectable at low magnification. Either in hairs pulled out by using some force at the beginning of the disease, or in those which fell out spontaneously at a later period, black, non-transparent masses were found, forming fusiform thickenings from 0.4 to 0.75 mm. long and over 0.1 mm. wide, embracing the whole thickness of the cortical and medullary substance. These black aggregations were found in the great majority (in over 90%) of the hairs on the body, even in the hairs of the eyebrows and the eye lashes, although here in a smaller percentage. The presence of thallium was not detected in these aggregations. This may allow us to assume that we are dealing with masses of melanin created in excessive quantity due to the catalytic action of thallium.

The deposits, he says, form at a relatively early stage, within the first 24 hours after the intake of thallium, and only in hairs which are still growing. Only

these therefore are subject to falling out as a consequence of the poisoning. The formation of deposits inhibits the growth of the hair, ruins the structure of the hair root and is most likely the cause of the shedding.

Sometimes deposits are present both in the root and in the shaft, a millimetre or so higher up, in which case repeated intake of the poison may be inferred.

We will not discuss the possible repercussions of the intoxication on the cardio-vascular system or on the urogenital functions. We will confine ourselves to a few considerations dealing with the respiratory function, the lines of Mees and the mental disturbances.

Breathing often becomes difficult on account of a toxic neuritis of the phrenic nerve and other branches of the cervico-brachial plexus, causing pain in the muscles of the trunk and along the intercostal nerves. Cyanosis sets in. This is why Prick insists on the great importance of resorting early to the iron lung. Many authors admit a polyneuritis of the visceral nerves, mainly of pneumogastric character, the vegetative nervous system participating sometimes in the process. This would explain the circulatory disturbances, which can be very serious.

Among the secondary disturbances, the lines of Mees (Figs. 11–13) deserve special emphasis. Mees, a Dutch clinician, was the first to describe them in arsenical intoxication. These lines, as one knows, are transverse, narrow and white, and progress towards the distal end of the nail as the latter grows. They also occur in thallium poisoning. According to Mazel, they appear 8 weeks after the beginning of the intoxication, according to Moeschlin, earlier, viz. after 4 weeks.

Fig. 11. Thallium poisoning, eighth week. The lines of Mees have appeared at the root of the fingernails.

The mental state of the patient sometimes deteriorates severely. Apathy and depression are followed by psycho-motor agitation. Total insomnia, which some authors claim to be of diencephalic origin, sets in from the the very beginning. Some authors speak of thallic encephalitis because of the analogy of the psychic disturbances with those of the lead encephalitis.

When reading the case reports one is struck by the fact that, in most cases, though the clinical picture was as complete as could be, the correct diagnosis was not reached. The explanation is that most practitioners are unacquainted with thallium poisoning. The fact that the classical treatises deal in a very limited way with the subject and that even specialized works on poisoning hardly mention thallium, is probably the major reason of this ignorance. Some physicians have a tendency to label as 'neurasthenia' or 'hysteria' any complicated syndrome for which they find no satisfactory explanation, and to lose interest in the case.

A typical example of an erroneous diagnosis (for which thallium poisoning was responsible) is given by the nearly incredible case of Schrader and Knorr.

The wife of a druggist became suddenly ill on 24 June 1926. From the beginning she complained of violent pain in the legs and chest. A few days later there was complete constipation, accompanied by abdominal cramps. The doctor attributed the complaints to pregnancy. A few days later, however, paresthesias of the lower limbs appeared. The woman had the feeling that her legs did not belong to her any more. Her condition deteriorated. The pain in the legs and feet became so agonizing that the slightest contact caused her to scream. She no longer slept at all, did not eat, and could hardly breathe. On 16 July she lost all her hair,

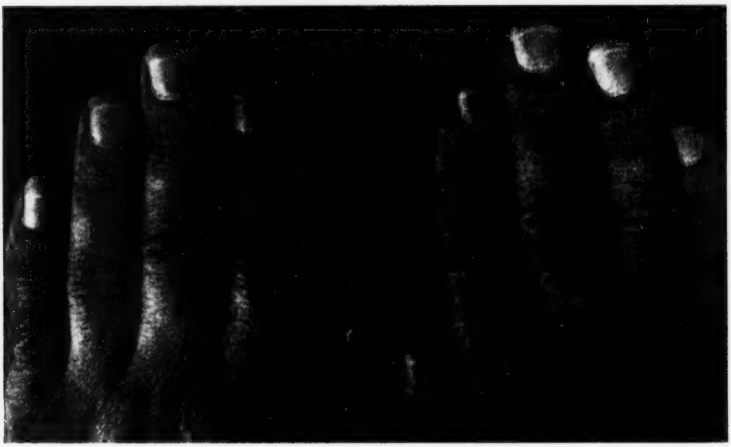


Fig. 12. Thallium poisoning, seventeenth week. The lines of Mees are now situated distally, near the nail margin.

including the pubic and axillary hair. The eyebrows, however, partly escaped. The family doctor diagnosed neurasthenia. About Christmastime, the patient felt better. She could walk a few steps. Her hair had re-grown about 2 inches. During the following 7 years, there were 8 relapses with identical symptoms. At the end of this period, however, and for the first time, the eyesight became poor.

A remarkable feature of this case is that this unfortunate woman was seen by several specialists (neurologists, surgeons, gynaecologists, physicians and lastly an ophthalmologist). All sorts of opinions were offered and there ensued tonsillectomy, extensive dental surgery, ovarian deficiency treatment, etc. She finally lost all hope and gave up further treatment. It must be emphasized that, each time she had to go to a hospital, a remarkable and quick improvement occurred in her condition. In the end it was discovered that her husband had surreptitiously poisoned her with thallium during all this time.

German authors, who have a thorough knowledge of thallium poisoning, consider the clinical picture as absolutely pathognomonic. A good deal, of course, could be said about the differential diagnosis. Lack of space, however, will not allow us to do so.

At post-mortem examination the condition of the hair requires the keenest attention. There is often hyperkeratosis of the soles of the feet. If the survival has been long enough, lines of Mees may be present. They are the expression of trophic disturbances and apparently do not correspond with deposits of thallium. The viscera show only slight changes. There may be slight fatty changes in the liver. The only finding in the chest is pronounced oedema of the lungs. The heart is free of pathological changes. The microscopic lesions of the nervous system are very extensive. They are pronounced in the nerves, the spinal cord and the brain and are degenerative in character. They can only be studied successfully, however, in the very rare instances where the post-mortem examination is carried out early after death. This is almost never the case in medico-legal practice.

Those interested in the neuropathological findings will find a detailed description in Prick's paper. Moeschlin has, more recently, described

Fig. 13. Thallium poisoning, seventeenth week. The lines of Mees are half-way up the nails of the toes.

severe degenerative alterations in the sympathetic chain, which would account, as we have said before, for some of the visceral disturbances.

Special emphasis must be laid on the fact that thallium salts, being very soluble, in contradistinction to arsenic, are very quickly eliminated. This elimination can even be considerably hastened by appropriate treatment. The consequence is that the toxicologist is faced with difficulties which he will only underestimate at his own risk. Many cases are only discovered belatedly, sometimes months or even years after the poison has been administered. If the victim is alive, the possibility of chemical proof is excluded. The task of the expert will be made easy if the poisoner has confessed, but a confession can always be retracted.

All authors are agreed that thallium is eliminated within a few days to a few weeks, depending on the amount taken in. Merkel gives us valuable figures:

Three children suffered from tinea capitis. Treatment by means of tablets of thallium acetate was administered. They received in a few days respectively a quarter, a sixth and a fifth of a gramme of the compound. They died respectively 13, 14 and 17 days after the start of the therapy. Toxicological examination revealed the presence of thallium but did not allow an assessment of the quantity, so small were the amounts remaining in the bodies.

Holzer tells the story of a young man who, on 4 June, attempted to commit suicide by swallowing 0.75 g. of thallium nitrate. The ensuing clinical picture was that of subacute thallium poisoning, which we have just described. On 2 July, 29 days later, thallium was still detected in the blood and the urine; 10 days later, however, there was none.

All the cases in the literature, including our own, are in conformity with the aforementioned findings. In lethal cases, the crucial point is the time of survival. When it is short or when the poison was administered until shortly before death, there is no difficulty. Thallium can even be detected histologically in the liver.

When death does not occur soon, and when survival has lasted, let us say a few weeks, the poison may have been eliminated to such an extent that it is no longer detectable by the classical chemical methods. It is advisable to resort to spectrography and polarography. Weinig and Philippow worked out the appropriate technique as far back as 1943, in connexion with a criminal case. All that was left for them to analyse were a few fragments of calcinated human bone. They nevertheless succeeded in their efforts. Their method has been applied successfully in Belgium more recently. We will not enter into details concerning amounts to be expected. Those interested are referred to the recent papers from this laboratory.

R. Michon and R. le Breton, in 1954 (*Annales de Médecine Légale et de Criminologie,* **34,** 136) described a radioactivation procedure for thallium, using a nuclear reactor. More recently, A. Heyndrickx [*Verhandelingen Koninklijke Vlaamse Academie voor Geneeskunde van België,* 'Toxicologie van het Thallium' (English summary) **19,** 333 (1957)] simplified the method, using the thallium spectrum in conjunction with a pulse-height analyser.

The radio-activation analysis may be applied both qualitatively and quantitatively, and is at present the most sensitive one.

The different methods used in chemistry, polarography, voltametry and spectrography for the detection of thallium in organs are reviewed in Heyndrickx's paper.

Poisoners nearly always resort to Zelio paste and not to Zelio grains. The paste is sold in tubes. The tubes contain 30 g. of paste or a little more. This paste contains thallium sulphate at a 2% concentration. One tube therefore contains from 60–70 ctgr. of thallium sulphate. The lethal dose being from 1 to 2 grammes, the ingestion of more than 1 tube is necessary to kill an adult. The paste, we know, is tasteless and does not smell. It can readily be mixed with any food without the suspicions of the consumer being awakened. As it is sold freely on the Continent of Europe and therefore readily accessible, the danger it represents is not to be underestimated, the more so as the clinical picture it brings about still nearly always escapes recognition. This is amply proved by the case reports recorded in this paper.

References

The references on thallium poisoning already exceed 1,000 in number. Matthys has surveyed those of special medico-legal interest in his paper *Neuf Observations d'Empoisonnement Criminel par le Thallium* in *Annales de Médecine Légale et de Criminologie*, **35,** 237–275 (1955). The whole subject has further been dealt with exhaustively from the clinical point of view in the excellent monograph by J. J. G. Prick, W. G. Sillevis Smitt and L. Muller, Ed. Elsevier Publishing Co., 1955.

Notes and News

First South African Medico-Legal Congress

The First South African Medico-Legal Congress was held under distinguished auspices in Johannesburg from 31 July to 2 August 1958.

It was sponsored by the South African Medico-Legal Society and over 60 members registered.

Patron: The Hon. Mr. H. A. Fagan, *Chief Justice*

President: Prof. S. F. Oosthuizen, *President of the South African Medical and Dental Council*

Vice-President: The Hon. Mr. L. Greenberg

Honorary Organizing Secretary: Dr. H. A. Shapiro

The *Programme of Sessions* was as follows:

THURSDAY 31 JULY 1958: 8.15 P.M.

Subject: 'Artificial Insemination.'
Speakers: Prof. H. R. Hahlo; Dr. F. Daubenton; Mr. A. Suzman, Q.C.
Chairman: Prof. S. F. Oosthuizen.

FRIDAY 1 AUGUST 1958: 9.30 A.M.

Subject: 'Symposium on Sudden Death in Infancy.'
Speakers: Dr. Seymour Heymann; Dr. S. Grové.
Chairman: Prof. I. Gordon.

11.15 a.m.

Subject: 'Problems of Blood Transfusion.'
Speaker: Dr. M. Shapiro.
Chairman: Dr. R. Turner.

2.15 p.m.

Subject: 'Problems of Consent to Medical Procedures.'
Speaker: Mr. M. A. Millner, B.A., LL.B., B.C.L. (Oxon.).
Chairman: Mr. A. Suzman, Q.C.

4 p.m.

Subject: 'Symposium on Acute Alcoholism.'
Speakers: Dr. Andrew C. Watt; Dr. R. Turner; Dr. P. R. v. d. R. Copeman.
Chairman: Dr. H. A. Shapiro.

8.15 p.m.

Subject: Two sound films entitled 'The Medical Witness' and 'The Doctor Defendant.' The screening was followed by a discussion on the films.
Chairman: Mr. I. Maisels, Q.C.

SATURDAY 2 AUGUST 1958: 10 A.M.

Visits to the South African Blood Transfusion Service and to the Medico-Legal Laboratories, Johannesburg.

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The Second South African Medico-Legal Congress

The outstanding success of the First Congress in Johannesburg ensured the continuation of these meetings.

The Second Congress will be held in Durban during the first week of July 1960, in association with the jubilee celebrations of the 50th Anniversary of the University of Natal.

Prof. I. Gordon (Dean of the Faculty of Medicine) is the Honorary Organizing Secretary. Further information can be obtained by writing to him at: The Medical School, Umbilo Road, Durban, Natal.

The Second International Meeting in Forensic Medicine

Following the First Meeting in Brussels in 1957, it was agreed that the Second Meeting would be held in 1960.

The probable venue is New York and those interested in obtaining further information should write to:

Dr. F. C. Camps,
Department of Forensic Medicine,
London Hospital Medical College,
Whitechapel,
London, E.1, England,

or

Dr. Milton Helpern,
Chief Medical Examiner of the City of New York,
125 Worth Street,
New York 13, N.Y., U.S.A.

British Association in Forensic Medicine

President: Dr. Francis Camps, The London Hospital, E.1.
Secretary: Dr. Donald Teare, St. George's Hospital, S.W.1.
Treasurer: Dr. Gilbert Forbes, The University, Sheffield 10.
Past Presidents: Sir Sidney Smith; Prof. John Glaister.

This Association was founded in 1950 as the British Association of Forensic Pathologists. The name was changed to its present form in 1956.

The Association meets twice a year, generally at the end of June or the beginning of July in the Provinces and at the end of November in London.

The meetings take the form of a Symposium in the morning and Short Papers in the afternoon, with a dinner in the evening.

An annual subscription of 2 guineas is paid by members resident in the British Isles and of one guinea by foreign members.

Meeting at Sheffield on 6 July 1957

Symposium: Death on the Table

Morning Session

Professor Glaister: This morning the meeting is taking the usual form of a symposium and the title is *Death on the Table.* It was felt some time ago, when the question of subjects was under consideration, that this topic was due for ventilation. The contributors were to include Professor Green, but unfortunately I am informed that he is unable to be with us so we are left with 4 four main contributors to the principal subject—viz. Dr. M. Platts of Sheffield, Dr. W. Griffiths of Sheffield, Dr. K. Mant of London and Dr. A. Hunt of Bristol, who are all well known to you. We do not intend to make a substitute for the unfortunate non-appearance of Professor Green but this will give us a little further latitude for discussion. It would be difficult to keep a subject such as this within the confines of minutes; therefore I welcome this extra breathing space and I do hope that following these contributions there will be a widespread contribution from the floor.

The Dangers of Carbon Dioxide Retention in Patients with Chronic Chest Disease

Dr. Margaret Platts, J. G. Graves Research Fellow, Department of Medicine, University of Sheffield*

Mr. Chairman, Ladies and Gentlemen: As I am neither an anaesthetist nor a surgeon I propose to confine my remarks almost entirely to observations made on conscious people. However, I hope you will agree that they are pertinent to the problems and dangers of anaesthesia in patients with chronic chest disease.

The particular group of patients I wish to discuss is that in whom respiratory function is impaired in such a way that carbon dioxide is retained in the body and the tension of this gas in the blood is increased. With a few rare exceptions these patients are either severely emphysematous or kyphoscoliotic. Since carbon dioxide combines with water to produce carbonic acid, a state of respiratory acidosis ensues. This is partially compensated by a secondary rise in the bicarbonate content of the plasma. The condition of respiratory acidosis can obviously be more precisely diagnosed by direct measurements of the pH and carbon dioxide tension of the arterial blood. In the absence of facilities for making these measurements, the simple finding of a high bicarbonate content or alkali reserve of the plasma will indicate the presence of respiratory acidosis in an emphysematous or kyphoscoliotic patient, if no other cause for electrolyte derangement coexists. However, the patterns of disease in which carbon dioxide retention occurs are so well defined that its presence may be predicted with a fair degree of confidence even when all biochemical measurements are impracticable.

The presence of a raised tension of carbon dioxide in the arterial blood indicates that alveolar ventilation is inadequate and consequently these patients are also invariably anoxic and cyanosed. Fig. 1 shows the arterial blood gases of a series of patients with chronic bronchitis and emphysema. The individuals represented by black dots on the graph were also suffering from pulmonary heart failure. As the oxygen saturation of the blood falls, the carbon dioxide tension rises. Moreover, patients with pulmonary heart failure have almost always a high tension of carbon dioxide in their arterial blood. The same general observations apply to a series of patients with severe kyphoscoliosis which I studied in collaboration with Dr. Hanley (Fig. 2). These patients whose arterial blood gases are shown in Fig. 2 were

* In receipt of a grant from the Medical Research Council.

not emphysematous, but those patients whose arterial blood was not fully saturated with oxygen also had carbon dioxide retention. Other diseases which produce cyanosis, such as valvular or congenital heart disease, are not associated with carbon dioxide retention.

Chronic respiratory acidosis induces various interesting physiological changes. Firstly, these patients are not usually dyspnoeic at rest, in spite of the presence of tensions of carbon dioxide in their blood which would make a normal person grossly breathless. It appears that the respiratory centre in these patients is insensitive to the normally stimulating effect of carbon dioxide. Various groups of workers have shown that inhalation of 5% carbon dioxide in air does not stimulate the respiration of patients with chronic respiratory acidosis as much as it does that of persons with a normal tension of CO_2 in the blood.[1, 2]

Inhalation of 100% oxygen by patients with chronic respiratory acidosis restores the oxygen saturation of the blood to normal and cyanosis disappears. However, occasional patients treated in this way become comatose and may die.[3,4] The onset of coma is associated with a gross rise in the CO_2 tension in the blood, and coma may in fact be due to the narcotic effects of high concentrations of this gas. Since the respiratory centre is relatively insensitive to CO_2, it seems that the stimulus to respiration is largely due to anoxia. Hence abolition of anoxia precipitates further respiratory depression. Fig. 3 illustrates this effect in short experiments on emphysematous patients.

The respiratory centre of patients with chronic respiratory acidosis is abnormally sensitive to the action of narcotics. There are numerous reports in the literature of emphysematous or kyphoscoliotic patients who died after administration of 1/12 to ¼ gr. of morphia.[5] Even barbiturates in normal doses may cause dangerous respiratory depression. Dr. Forbes asked me to give this paper as a result of a discussion we had about a

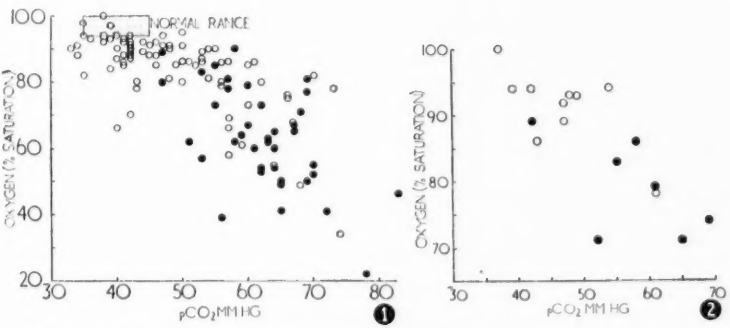

Fig. 1. Arterial blood gases of severely emphysematous patients.
Fig. 2. Arterial blood gases of patients with severe kyphoscoliosis.

kyphoscoliotic patient who was found dead in bed and in whom autopsy revealed no cause of death. This patient had taken a small dose of barbiturate the previous evening. I have seen another patient, an emphysematous woman, admitted to hospital in coma after taking 3 grains of butobarbitone. She was deeply cyanosed and almost apnoeic, but recovered consciousness within 10 minutes of an intravenous dose of aminophylline, which stimulated her respiration. Her arterial blood gases (illustrated in Fig. 4) show the respiratory acidosis and anoxia present on admission to hospital and the almost normal blood gases 2 weeks later.

Respiratory acidosis, like other forms of acidosis, produces a rise in the concentration of potassium in the plasma. The plasma potassium concentration in patients with chronic respiratory acidosis in whom the CO_2 tension in the arterial blood rarely exceeds 70 mm. Hg is slightly but significantly higher than normal. Brown has shown,[6] in experiments on dogs inhaling a mixture containing 20% CO_2, that the increase in plasma potassium concentration is progressive and after several hours reaches levels of 8–10 mEq./L. Moreover, he has demonstrated that the animals frequently die with ventricular fibrillation immediately after the CO_2 is with-

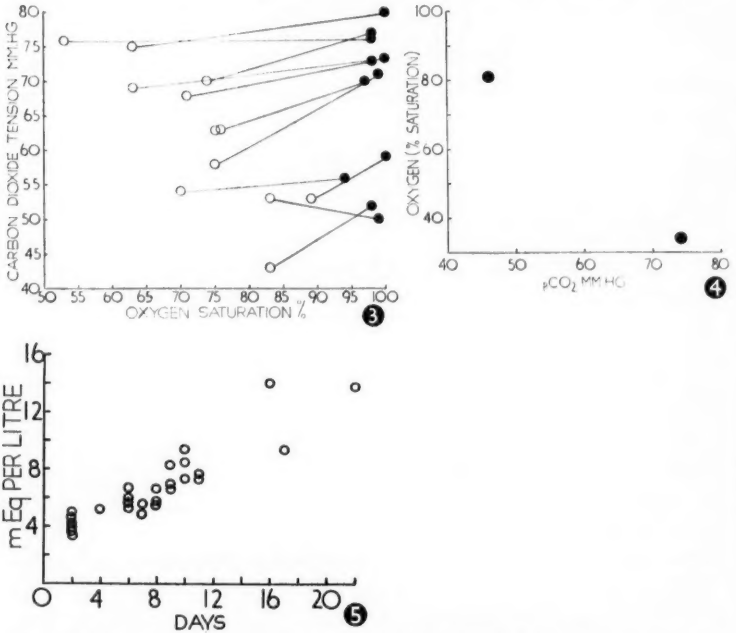

Fig. 3. Effect of breathing oxygen on the arterial blood gases of emphysematous patients.
Fig. 4. Coma with respiratory depression after 3 gr. butobarbitone.
Fig. 5. Change in plasma potassium concentration with length of time blood is stored.

drawn. Fatalities are associated with high concentration of plasma potassium. In recent reports carbon dioxide tensions of over 100 mm. Hg in the arterial blood have been found frequently during human anaesthesia and especially in operations on the open chest.[7] CO_2 tensions of this order correspond with those produced by the inhalation of 12% carbon dioxide and are therefore probably associated with a considerable rise in plasma potassium though, so far as I am aware, this has not been demonstrated during anaesthesia in Man.

It is not generally realized that the concentration of potassium in stored blood may be as high as 14 mEq./L. if the blood is nearing its expiry date (Fig. 5). Slow transfusion of blood is not associated with any rise in plasma potassium concentration in the patient, but such changes have been demonstrated during exchange transfusion in babies. It seems possible that rapid transfusion of old blood to a patient with respiratory acidosis is dangerous. As a shock-like state may supervene at the end of anaesthesia which has been associated with carbon dioxide retention, I think some fatalities may have been precipitated by the hasty infusion of blood in these circumstances.

From what I have said it appears that particular hazards accompany anaesthesia in any patient with chronic respiratory acidosis. In the first place, the usual methods of premedication may produce dangerous inhibition of respiration, while administration of oxygen will almost certainly produce a further increase in carbon dioxide retention. Since the patient's respiratory centre is insensitive to carbon dioxide, this increase in tension does not stimulate respiration and attempts to do so by inhalation of carbon dioxide mixtures are useless and dangerous. Since both administration of narcotics and of oxygen can produce coma in the unanaesthetized emphysematous patient, it is hardly surprising that after anaesthesia these patients may fail to regain consciousness. I have seen a severely emphysematous patient operated on for removal of a pulmonary cyst fail to breathe spontaneously for over an hour after cessation of anaesthesia. He had been premedicated 3 hours previously with $\frac{1}{4}$ gr. morphia and anaesthesia had included administration of oxygen and curare. Administration of neostigmine did not restore respiratory movements but spontaneous respiration occurred 10 minutes after an injection of nalomorphine and substitution of room air for oxygen as the inhaled gas.

The dangers of carbon dioxide retention during anaesthesia of the patient with normal lungs are well known and I do not propose to discuss them. However, fatalities due to respiratory acidosis in normal persons may be more frequent than was realized until quite recently. The recent correspondence in the *British Medical Journal* suggests that the syndromes hitherto known as cyclopropane shock and neostigmine-resistant curarization may, in fact, be the result of severe carbon dioxide retention.

References

1. Scott, R. W. (1920): Arch. Int. Med., **26**, 544.
2. Alexander, J. K., West, J. R., Wood, J. A. and Richards, D. W. (1955): J. Clin. Invest., **54**, 511.
3. Comroe, J. H., Bahnson, E. R. and Coates, E. O. (1950): J. Amer. Med. Assoc., **143**, 1044.
4. Westlake, E. K., Simpson, T. and Kaye, M. (1955): Quart. J. Med., **24**, 155.
5. Roussak, N. J. (1951): Lancet, **1**, 1156.
6. Brown, E. B. (1955): Proc. Soc. Exp. Biol. Med., **90**, 319.
7. Ellison, R. G., Ellison, L. T. and Hamilton, W. F. (1955): Ann. Surg., **141**, 375.

Unexpected Emergencies Under Anaesthesia

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Gentlemen: I am very honoured to be called upon to address this meeting and I have a feeling now of what the early Christians felt like when they were thrown to the lions. The title of my communication automatically excludes, I think, the hazards of defective or mis-applied apparatus, the difficulties of patients who have known disturbances of function, the accidents occurring from and associated with careless or improper administrations, the hazards of blood transfusion, or major surgical mishaps; so that you can see, after being deprived of the common and therefore, I suppose, what you might call the 'expected' disasters occurring during surgical procedures, I am forced to lead you into rather small print stuff; and I hope you will forgive me if I tread these exotic paths rather delicately.

I will use the words 'apparently healthy' when enunciating my brief. I do this because I firmly believe that the physiologically healthy do not collapse or die under anaesthesia without cause. Those who do die or suffer severe collapse under anaesthesia, without pre- or post-mortem signs of disease, do so largely because of the action of the drugs administered to them or the way in which they are administered; and whilst the clinicians are always very grateful of the 'No evidence that the anaesthetic was anything but skilfully administered' type of report, I think we must recognize that possibly we speak with our tongues in our cheek. But I have no intention here of stealing Dr. Hunt's thunder. Also I want to make it clear that, as far as I know in my limited experience, there are no *unexpected* emergencies or disasters under local anaesthesia which are functions of local anaesthesia *per se*. Indeed, I suppose that if one goes on long enough most of the emergencies of local anaesthesia will happen sooner or later, but as they are all both clear-cut and expected, I do not think they enter my orbit to-day. Persons may be 'apparently healthy', I think, on three counts. First, the pre-anaesthetic examination may have been deficient and the clinical history or physical signs may have been missed or misinterpreted. Most pre-operative examinations are done by the house staff and a good deal depends on their thoroughness and their appreciation of the anaesthetist's point of view. It would perhaps be interesting on some other occasion to consider in what different ways the anaesthetist views the patient than from a strictly surgical aspect. Moreover, with the increasing complexity of anaesthetic techniques and methods we are probably now, and certainly in the future, asking too much of the newly qualified to be able

to examine and report on cases for anaesthesia, particularly where considerations of *choice* of anaesthetic are concerned. Simply as an illustration I would like to review my cases of the last 8 months.

Of a total of 415 cases, 156 were reported as having 'trouble', although on investigation 32 of these were not regarded as significant by the anaesthetist. However, in 23 other cases (0.5% of the total) the House-surgeon's report was either misleading or deficient. In 5 of these 23 significant information was suppressed and in the remaining 18 a significant history or physical sign had been missed, although in only 4 of these 23 cases would the lack of information have possibly led to catastrophe at operation. This demonstrates particularly well the law of diminishing returns; but it is possibly going to be of increasing importance, particularly in large centres where the proportion of difficult and complicated work is generally higher, that cases should be seen by the anaesthetist concerned in the pre-operative period. Of course there are more valuable aspects of the anaesthetists' pre-operative visits, but it is not my function to discuss them here; nor, I think, need I discuss the organization or financial problems of much complexity that such an innovation would raise.

There is yet a second type of patient in whom a catastrophe occurs without *adequate* post-mortem signs to account for it. We clinicians are perhaps too prone to breath a slight sigh of relief when an autopsy reveals some basal collapse or early bronchopneumonia or a little coronary atheroma, or one kidney . . . or something of that sort. The tendency to consider such findings to be the real cause of death must be firmly resisted. It is in such cases that the only people who are likely to know or adduce the real cause are those who attended him during life: much closer contact between clinician and pathologist is to be desired in such cases; closer, that is than the rather stilted note which often passes to the Coroner's Officer. Many of us feel that the official legal enquiry is not always the best way of advancing knowledge in this class of death and a less formal and more confidential exchange of views in that direction is sometimes of more value.

In the third group of cases, that of the emergencies arising from undiagnosed disease in the 'apparently healthy', the pathologist may solve the problem for the anaesthetist, or the anaesthetist may, in the light of his clinical observations, give a valuable clue to assist the post-mortem examination. Undiagnosed disease (notwithstanding a careful routine pre-anaesthetic examination) of sufficient severity to cause death or near death is uncommon and the reports about them are therefore only sporadic in the literature. The adrenal cortex seems to be the chief offender. Adrenal cortical depletion with a lack of response arises, of course, in patients with long continued cortisone treatment where this drug is suddenly withdrawn; but although the fatal outcome here is usually post-operative, a severe and intractable hypotension resulting in death may occur under the anaesthetic itself, particularly when the operation is prolonged and severe. The lia-

bility at this stage, incidentally, as has been recently shown, can still occur up to 6 months following the cessation of cortisone treatment. I have records of 5 cases of undiagnosed Addison's disease, all ending fatally on the table. All of them had normal pre-operative blood pressure readings, but all entered a phase of hypotension and peripheral circulatory failure barely reversed by pressor agents and transfusion with no sustained improvement and eventual death. As an example, a male patient aged 47, was diagnosed as a case of chronic duodenal ulcer. Pre-operative examination revealed a blood pressure of 120/70 mm. Hg and the rest of the clinical examination and history were unremarkable. After induction the patient suddenly went ashen with no recordable pressure. Pressor agents had no effect. The apex beat was just detectable and exploration of the abdomen revealed bilateral adrenal tumours. Intravenous blood, plasma, l-noradrenaline, hydrocortisone and saline had no effect and the patient died 2 hours later. Autopsy showed both adrenals completely replaced by tumour. In this group of adreno-cortical suppression I would like to know where I may now put the status thymo-lymphaticus. Perhaps you will give me your ideas on this.

Undiagnosed phaeochromocytoma should be considered whenever a patient, particularly aged between 20 and 40, previously apparently healthy, suddenly develops severe hypertension and tachycardia. As an example we may quote a female patient aged 44, with a pre-operative blood pressure 150/90 mm. Hg, scheduled for a cholecystectomy. During the abdominal exploration the blood pressure soared right off the scale and the pulse was hardly countable. Despite various measures the blood pressure never fell until ventricular fibrillation and death occurred. There was a left adrenal tumour.

The mode of presentation of phaeochromocytoma causing acute pulmonary oedema with left heart failure occurred in a case which came under my charge. A man aged 34 with a pre-operative blood pressure of 140/90 mm. Hg, was again ' apparently healthy ' with no complaints. There were no signs or symptoms of disease apart from a small apical shadow. Five minutes after induction of general anaesthesia for bronchoscopy there was a sudden onset of severe pulmonary oedema with a profuse outpouring of fluid, complete cyanosis, extreme tachycardia and a peripheral venous collapse. Working fairly quickly a blood pressure cuff was applied (250/180 mm. Hg) and immediate treatment with phentolamine and mechanical measures to deal with the pulmonary oedema to improve the patient's ventilation resulted in a live patient after a very sticky 45 minutes. Subsequently a small pheochromocytoma was removed from the left side bifurcation.

In a third case of undiagnosed phaeochromocytoma, sudden cardiac arrest occurred, presumably due to ventricular fibrillation. In this case trichlorethylene was used and it is possible that all the other anaesthetic agents that

sensitize the myocardium to the action of adrenaline also carry this risk. As in Addison's disease the pre-anaesthetic blood pressure may show little of note and, in fact, it may be impossible in clinical practice to investigate all patients who come to operation with a blood pressure of the order of 140/90 mm. Hg. The significant small clues in the history may be glossed over or completely missed even by the most expert and in my collection of cases of undiagnosed phaeochromocytomata (only 5 have been reported in the literature) 9 have died on the table. In passing, we may perhaps note in this context the possible association of adrenal manipulation with sudden cardiac arrest during nephrectomy, particularly when agents which sensitize the myocardium are employed.

We have here demonstrated a tachycardia and a rise in blood pressure following adrenal manipulation under anaesthesia with induced hypotension with hexamethonium, suggesting a release of adrenal medullary hormones. I had the task of looking through some investigations into anaesthetic deaths and discovered that 5 nephrectomies had died quite suddenly for no apparent reason on the table. Sudden cardiac arrest was the 'cause of death', but in all these cases it was interesting to note that the anaesthesia was either cyclopropane or trichlorethylene and in 4 of them the operation was difficult, involving a lot of manipulation and pulling and stretching. This is pure hypothesis and experimental confirmation obviously has to await some suitable method of establishing the catechol amines in the blood.

Unexpected Pulmonary Emergencies under Anaesthesia. The most important, because it is about the only one for which anything can be done, is unexpected tension pneumothorax. This occurs a little more frequently now with the advent of positive-pressure ventilation. However, under anaesthesia the clinical picture is not at all clear-cut, but it should perhaps come to mind when unexplained cyanosis occurs in spite of an apparently clear airway and full ventilation. The diagnosis in this early stage may be difficult. I have known two M.R.C.P.'s, 3 F.R.C.S.'s and 2 F.F.A.'s walk away from a patient shaking their heads about a supposed cerebro-vascular accident in a man of 38 who had just had a *right* upper lobectomy performed. Auscultation of the heart sounds just before E.C.G. to exclude a coronary occlusion revealed the heart sounds as distant and a needle in the *left* chest restored oxygenation and resolved the signs of 'cerebral shock'. A percussion note under the conditions of the operating table is unreliable and a portable X-ray of the chest should be called for at once, even on suspicion of this condition. The condition is certainly lethal under anaesthesia unless recognized and properly treated.

Pulmonary embolus may occur under anaesthesia. Clot embolus has occurred in my experience once during the induction of anaesthesia in a patient described as on routine clinical examination as 'quite healthy'. I hurriedly checked the oxygen supply in my machinery, until the cyanosis, hypotension and tachycardia in the presence of an undoubtedly clear airway

and full ventilation, rang somewhere the right bell. Other unexpected forms of pulmonary embolus are fat, tumour and amniotic emboli and in these the syndrome of bronchial shut-down nearly always occurs. The bronchospasm is usually severe and fails very often to respond to any direct treatment, the patient dying soon afterwards. The amniotic embolus is perhaps the most potent in this respect. Bronchial shut-down may also occur where neither the bronchi nor the pulmonary vascular bed are affected. I illustrate this with the case of a woman of 42 years with a mass in the left chest thought to be a fungus infection. At thoracotomy it was found to be a rather soft greyish tumour invading the left atrium. Removal was attempted and as the left main bronchus was divided early it had to be persisted in even when it became quite clear that the case was hopeless. About 15 minutes after the left main bronchus was divided there was a complete obstruction, so extreme that my first thought was retraction of the remaining bronchus. Secretions were not present and it was then observed that the heart had stopped. Massage was unavailing and post-mortem examination showed tumour embolism; the carotids, the left ventricle, the aorta right down to the bifurcation were full of this soft greyish tumour which, incidentally, was later found to be possibly of primary adrenal origin: for this case also had persistent hypotension which was very difficult to correct. The liver and pelvis were both full of tumour and both adrenals were replaced by what was probably a primary adrenal tumour.

Vascular accidents, both cerebral and coronary, seem to be very uncommon on an *unexpected* basis. As far as cerebro-vascular accidents are concerned we may note two things. First, a prolonged Trendelenburg position may initiate a cerebral thrombosis (which is not very surprising) and a thrombosis may occur under anaesthesia, the patient recovering consciousness and a certain amount of re-activity, although slowly, before relapsing into coma and death, so that the recovery of consciousness does not exclude the possibility of the thrombosis having occurred during anaesthesia. There are only two cases in my files of this.

The last class of patients with undiagnosed and unsuspected disease is those with unsuspected arterio-venous shunts, either systemic or pulmonary. These are not very common. Most arterio-venous shunts are readily recognizable, but when they exist they may also, especially in association with intravenous barbiturates, produce an alarming hypotension and even death. In my experiense an increasing number of patients is taking potent drugs on a maintenance regime and catastrophies may occur from the potentiation of such drugs by agents used in anaesthesia, sometimes of course helped by extremes of posture. For example, the hypotensive agents, particularly the combination of the ganglion-blocking agents and Reserpine, and some of the tranquillizing drugs, may result in an uncontrolled and unrestorable hypotension, when even small doses of intravenous barbiturate or intravenous pethidine are employed. An abrupt with-

drawal of some drugs unknown to the anaesthetist may be of great importance. Only 2 weeks ago we managed to forestall the sudden stoppage of hydrocortisone in a patient due for operation 2 days later. However, as more potent drugs are made available and are becoming more popular, it seems reasonable to me that the pre-anaesthetic examination should now include a routine question to determine whether the patient has been taking any drugs and if so, which.

In a somewhat lighter vein I would remind you that the patient is not the only person at risk in the operating theatre—the staff may also suffer, and rather dramatically at times. As this is a meeting of what the *Daily Express* would, I believe, call 'medical detectives', perhaps I may finish with a true story, at the same time assuring those present that unless the Treasurer has an undisclosed surplus, there is no prize for the first correct result. The time is a warm summer's day in 1949; the place Israel. (There is no particular significance in the place; the accident could have happened in other parts of the world). Operating that day finished at 2.30 p.m. At 4.30 p.m. a nurse began to clean the glass top of a Boyle's anaesthetic machine, using a spirit rag, as was customary. There was a violent explosion. The anaesthetic room was a shambles and the nurse was dead. Investigation of the wreckage showed that a cyclopropane cylinder had exploded, scattering chunks of cylinder casing around the room in shrapnel fashion. Furthermore, all the control valves on the anaesthetic machine were fully open, but the cyclopropane cylinder valve was firmly and tightly shut. The theatre was ventilated but not air-conditioned and the inside temperature was 80–82° F. It was not the practice in the theatre to wear conductive footwear. The anaesthetist, a man of uncommon perception, went away to think; when he returned he interviewed separately every member of the theatre suite: asked each one of them one question and one question only. It was always the *same* question. At the end of the interviews he indicated one member of the operating theatre staff and said: 'You were *really* the cause of that explosion'. What was the question the anaesthetist asked?

I would like to thank you, *Mr. President,* for the courteous way in which your members have received my communication, and at the same time I would like to acknowledge the help of a number of colleagues and friends whose brains and experience I have unashamedly picked to provide me with the material I have presented to you to-day.

Immediate Post-Operative Deaths

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The field of immediate post-operative deaths has very indeterminate boundaries, and I have confined the cases which I shall discuss to fatalities which occurred, or which might well have occurred, within a short time of operation. I am not restricting myself entirely to those cases where death occurred within 2 hours of operation, but I am including those which could well have occurred during this time, but perhaps fortuitously occurred somewhat later. All the selected cases are those of death apparently arising out of surgical or anaesthetic errors, lack of technique, experience or management, and the maladministration of intravenous fluids, including blood. I have excluded all deaths which have resulted from the presence of, or a sudden extension of, natural disease, whether or not this had been contributed to by the operation or anaesthetic. Thus, having excluded the most common causes of immediate post-operative death, I shall deal with the unusual but nevertheless ever-present potential dangers which beset the patient during his immediate post-operative period.

I have selected 43 cases from some 15,000 Coroners' autopsies of all types; 8,000 of them are cases of Dr. Simpson's, the remaining 7,000 are my own. There are also 23 cases of massive collapse of the lungs which have come entirely from my personal cases.

In order not to overrun the 15 minutes allotted to this paper, I only intend to go into certain cases in detail, merely giving the frequency of the remainder in this series.

I have divided the causes of death into 3 groups:

i. Post-operative surgical mishaps;

ii. Post-operative anaesthetic mishaps;

iii. A combination of the two.

Under post-operative mishaps I class, firstly, massive secondary haemorrhage due to a slipped ligature, or unligated major vessel, or simply poor general haemostasis.

Twelve such cases occurred, and were made up as follows: Four cases of ligatures slipping from a major vessel, in each case the pulmonary artery, following pneumonectomy for carcinoma. Three cases of failure to ligate a major vessel, one of which was the unrecognized perforation of the uterine artery by a dilator during a therapeutic abortion.

One each of the following cases:

Accidental excision of the femoral artery during a Trendelenburg operation for varicose veins;

The accidental puncture of a coronary vein during paracentesis;

Poor general haemostasis following thoracotomy (thoracic sympathectomy and oesophagectomy for carcinoma);

Secondly, I include the unrecognized accidental perforations of a viscus. Three such cases occurred, and each involved the perforation of the bladder. The operations were those of cystoscopy, herniotomy and punch prostatectomy.

Thirdly, I have grouped cases of air embolism together, and I found, all told, 7 cases.

Two of these occurred during blood transfusions. One of these occurred recently during the exchange transfusion of a newborn child for Rhesus incompatibility. In this case the catheter in the umbilical vein was frequently blocked by blood clot, necessitating constant withdrawal and the insertion of a fresh catheter. It is believed that air must have entered the circulation during one of these exchanges. Shortly before the infant died the doctor carrying out the transfusion withdrew a syringeful of air from the umbilical vein when attempting to withdraw blood.

A second fatality occurred during the refill of an artificial pneumoperitoneum, the air entering the circulation through a raw area on the surface of the liver which had been produced when some recent adhesions had been stripped by the increasing pressure of air in the peritoneal cavity.

Four cases occurred at the end of, or immediately after surgical operations. These operations were for thyroidectomy, mastectomy and lobectomy. The fourth was for an extensive skin graft following burns. In this case numerous small vessels were kept open by scar tissue, and air entered the circulation through these vessels.

The fourth group concerns 4 deaths due to bad errors on the part of the surgeon. These include the closure of the proximal end of a colostomy loop; the complete symmetrical bisection of the internal capsules during leucotomy; the mistaking of a volvulus of the caecum for an ovarian cyst.

The final case is worthy of more detail. It concerns a consultant gynaecologist who was treating prolapse in old women by sclerosing the vaginal wall by injections of 5% phenol in almond oil. One day he asked the theatre sister what she had besides phenol in almond oil, and on hearing that they had phenol and camphor B. P., which incidentally contains 80% of phenol, he decided to try it. The lady had a spasm on the table after the first 2–3 c.c. had been injected. Injection was stopped but the deceased died some hours later from phenol poisoning.

Finally, under this group of deaths I include the blood transfusion accidents. Air embolus has already been mentioned, and I do not intend to

discuss the transfusion of incompatible blood. The following fatality, however, is worthy of mention.

An elderly lady, aged 82 years, was found to have an inoperable carcinoma of the rectum with secondaries in the liver. As a result of this she had become anaemic. In October 1956 she was admitted to hospital for blood transfusion. Three pints of blood were cross-matched but only 2 pints were given. She was much improved and returned home. Five months later she was again rather anaemic, and was re-admitted to hospital for transfusion. A further 2 pints of blood were cross-matched. The first pint of blood was run in during the day, and the nurse went to the refrigerator and removed what she thought was the second bottle of blood, which was in fact the bottle of blood which was not used in October and which had not been returned to the laboratory but kept in the ward refrigerator. She checked the name on the bottle but not the date. The patient became jaundiced and died a few hours after the second bottle had been given.

With regard to death resulting from direct complications of the anaesthetic itself, post-operative deaths are rare. I found one case of idiosyncrasy to Brevidil, a short-acting relaxant. A patient in a mental hospital was given Brevidil before receiving his ECT, but never began spontaneous respirations. It is not entirely certain in this case whether the ECT or the Brevidil was responsible for the paralysis of the respiratory centre.

We then come on to the mechanical blockage of the airway post-operatively by vomit, apparatus or body fluids including blood. I have excluded cases of regurgitation of stomach contents following or during operations after accidents, as pre-operative preparation was often impossible.

Having excluded these, there are 3 cases in which death was due to the inhalation of vomit.

I only found one case in this series where the airway was blocked by apparatus—in this case, a dental swab.

There were 7 cases of respiratory obstruction by the inhalation of blood into the air passages following operation on the respiratory tract. Three of these followed tonsillectomy, and another 3 followed biopsies for malignant disease.

There were 2 cases of secretions collecting and causing collapse of the lungs, and there were 2 cases of acute post-operative hypotension due to adrenal insufficiency. In one case a middle-aged man was admitted as an emergency with acute appendicitis. It was known he was suffering from Addison's disease but he was thought to be adequately stabilized. Some 90 minutes after the removal of a gangrenous appendix he developed an acute hypotension and died.

The other case followed the removal of a phaeochromocytoma.

Another case of interest is that of acute vagal inhibition during the removal of a tracheal tube some 15 minutes after the completion of the operation.

We then come to the final group of cases where surgery and anaesthesia together appear to have played a part. These are the cases of acute post-operative shock and massive collapse of the lungs, the latter occurring towards the termination of the operation or sometimes several hours afterwards. My experience is that in many of these cases, but by no means all, both the surgeon and the anaesthetist had been of relatively junior status. One finds far more trauma in the operation field where junior surgeons carry out operations, and this must contribute. In the majority of cases of massive collapse of the lungs I have seen, the anaesthetist has been comparatively junior although no fault could be found with his technique, or the operation has been of a very extensive nature and of long duration. Among my personal cases, I have 23 deaths due to massive collapse of the lungs, where the collapse has not been contributed to by fluid in the bronchial tree. The ages of these patients range from 18 to 93 years, and cover almost every class of operation from simple lens extraction to major abdominal or thoracic surgery. In some cases it did appear that the anaesthetist might have been a little over-generous with the anaesthetic.

In one case worthy of special mention a woman aged 48 years was operated on at night by a House-surgeon for appendicitis; but, discovering a gangrenous appendix, he sent for the Registrar, and there was a half-hour delay before he arrived and continued the operation.

The number of cases I see are becoming fewer. Among the first 1,000 autopsies I carried out after the War there were 9 cases of massive collapse; there have only been 3 such cases among the last 3,000 cases.

I have seen other cases of massive collapse following general peritonitis, continuous narcosis treatment, ECT and insulin shock.

In this category one should include those difficult cases, from the pathologist's point of view, where an elderly and usually hypertensive person dies following a ' cold ' major operation, and autopsy and full enquiry with the anaesthetist and surgeon fail to elicit why death should have occurred. Again, in many of these cases both the surgeon and the anaesthetist are of junior status, but the surgery is technically perfect and there is no suggestion of miscarriage with the anaesthetic.

In conclusion, I should like to say that in my experience the quality of both anaesthesia and surgery has risen greatly during the last 12 years. Fewer and fewer general practitioners with surgical experience are undertaking major surgery, the surgery being carried out now by surgical consultants. The considerable number of operative and post-operative deaths which swelled my figures after the War were due to surgery and anaesthesia being carried out by less experienced general practitioners.

Anaesthetic Causes of Death on the Table

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I was greatly honoured to be asked to take part in this symposium, but of course respect is due to Bristol in any discussion on anaesthesia. The first research department in the world to investigate the therapeutic effects of gases was opened in Bristol in 1798 by Thomas Beddoes, and its first Director was Humphrey Davy. Soon after it was founded, nitrous oxide was first used there. Beddoes even administered the gas to his wife, and describes how she had 'exquisitely pleasurable sensations, a propensity to muscular exertion and could walk much better up Clifton Hill, and frequently seemed to be ascending like a balloon'. However, the Bristol Pneumatic Institute was short lived, and although Davy made a few vague references to the use of nitrous oxide for anaesthesia it was not used until some years later. So far as I can trace, we had no anaesthetic deaths at the Pneumatic Institute and, in fact, historically Dr. Camps has a clear precedence in this subject, as the first anaesthetic death on record, complete with Coroner's verdict, was at the Colchester and Essex Hospital in 1847. This was a man who died some hours after ether anaesthesia for lithotomy. The first death on the table attributed by a Coroner to the anaesthetic occurred during the administration of open chloroform to a young girl for the removal of a toe nail. This happened at Grantham in 1848. She was induced with a pad wetted with a teaspoonful of chloroform, and it would appear that she got rather light and collapsed suddenly, presumably from ventricular fibrillation. But an autopsy was performed and the cause of death was given as congestion of the lungs. Now that brings me to a topic which might well be discussed here to-day: what conclusions *can* justly be drawn from post-mortem appearances after death possibly attributable to anaesthesia? Firstly, the *external appearances* of the body are unlikely to be of much value. The colour of the hypostases and the fingernail beds is, I think, a very uncertain factor on which to form any idea of the oxygen saturation before death. If the patient was deeply cyanosed before death the body will usually remain cyanosed, unless it is refrigerated, when it will turn pink in no time. If the patient is pink when he dies the hypostases often become blue after death.

Respiratory System. In the respiratory system there are only a limited number of pathological appearances worthy of discussion in this context. On the surface of the lungs there may be petechiae; the substance may be collapsed, it may be oedematous, or it may be congested. There may be material in the bronchi.

Petechial Haemorrhages. There are a lot of things I do not understand about petechial haemorrhages, especially in the pleura. I am not even sure

which vessels burst to form them. I know they are capillary in origin, but are these capillaries from the bronchial or the pulmonary circulation, and what are the pressures in the pleural capillaries? I find it difficult to understand why petechiae are so frequently seen in the interlobar fissure and on the posterior surfaces. Are they an indication of hypoxia or anoxia? Can they appear after death?

The view that petechiae are indicative of a low oxygen saturation in capillary blood in life is becoming increasingly suspect, although one still hears them referred to as asphyxial, hypoxic or anoxic haemorrhages. One of the objections to the traditional view is that to consider capillary haemorrhages as caused by hypoxia presupposes that a low oxygen saturation has a direct effect on the permeability of the capillary endothelium, a view fairly generally accepted, but which now is becoming questioned. I am afraid I have not got time to go into detail, but one of the more direct pieces of evidence against the role of hypoxia is that if you perfuse a rabbit's hind limb through the artery with venous blood at arterial pressure, thus rendering just that limb of the animal hypoxic, while the other limb is normal, there is no increased fluid transudation in the hypoxic limb. When the animal is rendered oedematous in a variety of ways the anoxic limb always shows the same amount of oedema as the normoxic one.[1]

An alternative explanation of the occurrence of petechiae is a 'mechanical' one. In the thorax there are two major inter-related forces, the intrathoracic respiratory pressure and the intravascular pressure. The negative pressure in the chest can rise higher than one might think. One can voluntarily create a negative pressure of about 80 mm. Hg, and a positive one of up to 120 mm. or more. A negative pressure of this order may well predispose to capillary bursts, and presumably the greatest negative pressure will be exerted at the pleura. The second factor is probably more important, and a sudden rise in pulmonary capillary pressure may follow a rise in pulmonary venous and left atrial pressure, or a sudden increase in right ventricular output, and it seems possible that this sort of mechanism can be the cause of the frequent finding of petechiae in congestive cardiac death, and in sudden death from coronary artery disease. The common finding of petechiae in the interlobar fissure is, I think, some indication that increased pulmonary venous pressure plays some part in the production of pleural haemorrhages. I have noticed, when perfusing lungs with radio-opaque material through the pulmonary veins, that the first place a small vessel burst is likely to occur is in the interlobar fissure. If one perfuses the pulmonary veins of a cadaveric lung with blood under a fairly high pressure, petechiae appear quickly round the hilum and in the interlobar fissures.

I sometimes wonder if a few pleural petechiae can appear after death. It seems theoretically possible that blood can leak from capillaries after death. The blood remains fluid in small vessels indefinitely. If the

fibrinolytic mechanism after death enables the blood in larger vessels also to remain fluid, one can envisage at least a few centimetres of water pressure in the capillaries of the dependent part of the pleura. Furthermore a negative intrapleural pressure is often maintained after death and I have measured negative pressures up to −10 cm. H_2O in the pleura 2 days after death. If you passively fill a lung with blood after death at a low pressure of about 10 mm. Hg, after about half an hour a few pleural haemorrhages start to appear.

There are two other observations which may be relevant. It is not uncommon to see small blotches of haemorrhage in post-mortem lividity in the skin, especially when it is intense and the body has been lying on its face, and presumably these are of post-mortem origin. If one accepts this, one accepts that gravity alone can cause blood to leak from capillaries after death. Also I have several times observed a distribution of pleural petechiae, especially in babies found dead in their cots, which is very suggestive of gravitational influence: that is on the cardiac surface of the uppermost lung and the costal side of the lowermost. From the interpretational point of view I think one should regard a few petechiae as possibly having no significance, and a larger number as not necessarily indicating anoxia.

Pulmonary Collapse. The first problem to me is how far the appearance of the lung at necropsy reflects the degree of aeration before death. Presumably the lungs lose some air after death, especially if the chest has been opened, e.g. for cardiac massage; but not, I think, to the extent of complete airlessness, with the lung parenchyma sinking in water. If at autopsy massive pulmonary collapse is found, I find it often impossible to reach a conclusion as to the cause of atelectasis from the post-mortem findings alone. Lucas[2] considers that complete resorption collapse (i.e. collapse not due to pressure from the pleural space) does not occur in the absence of lower respiratory obstruction. He does not agree that there is any great likelihood of atelectasis if the inhaled gases contain a high proportion of oxygen, and the so-called 'nitrogen scaffold' is removed. He points out that if the bronchi are not obstructed, the absorbed oxygen is replaced passively by diffusion from the trachea. By the same token complete atelectasis will not result from underventilation if the bronchi are patent. If the bronchi are found at autopsy to be full of viscid mucus and there is massive collapse, one is even denied the conclusion that this was cause and effect. Collapse due to bronchospasm may be associated with the production of excessive amounts of viscid mucus, and therefore the presence of the mucus may be a purely secondary phenomenon. One possible reason for this is that both bronchospasm and excess mucus production may follow parasympathetic overaction. Procaine block of the vagus trunk will dramatically relieve bronchospasm in open chest operations and allow collapsed previously uninflatable lung to be easily reinflated; and parasympathetic overactivity is well known to be associated with the production

of unusually viscid mucus. Incidentally, it is very easy to inflate collapsed lung after death so that anaesthetists can easily remove evidence of atelectasis by resuscitative measures.

Before leaving bronchial obstruction, I must say a few words about the significance of the finding of stomach content in the air passages in patients dying on the table. Personally, whenever I have found stomach contents in the trachea or bronchi in operation deaths, there has always been clinical evidence that this had in fact occurred during life. However in the Association of Anaesthetics report last year of 1,000 anaesthetic deaths which included 110 cases clinically attributed to inhalation of stomach contents, in many (they do not give a number) nothing was found in the air passages at autopsy.[3] Also they point out the well known fact that the inhalation may be agonal and not causal, in anaesthesia as in other conditions.

Pulmonary Oedema. I am afraid it would take all my time to discuss the aetiological significance of pulmonary oedema. Suffice it to say that the evidence that pulmonary oedema can be caused by hypoxia is not conclusive; and that in patients who have been heavily transfused on the table the finding of pulmonary oedema does not necessarily, in my opinion, mean that they have been overtransfused.

Pulmonary Congestion. I do not think this means a thing in an average autopsy. That concludes what I have to say about the respiratory system.

The other systems do not enter so much into a discussion on death occurring during anaesthesia, which is the limit of my brief. Such a death may be due to cerebral anoxia, but one would not expect to find morphological changes unless the patient had survived for some time. Deaths from ventricular fibrillation or cardiac arrest do not produce any changes of themselves in the cardio-vascular system, despite the fact that I recently heard two pathologists discussing from the appearance of a heart whether it failed in systole or diastole! But there are two problems which must be considered in connexion with death due to the anaesthetic. They are the existence of status thymolymphaticus and the role of coronary artery disease.

Status thymolymphaticus seemed to have passed into the realm of medical mythology until fairly recently, after a good run of 300 years, but it now seems to be rearing its head again. This is partly because of the suggested role of adrenal insufficiency in the general adaptation syndrome, and the well-known fact that in gross suprarenal insufficiency the thymus and lymphatic tissue are hypertrophied, although I do not see how that affects the position at all. In case you have not seen it, I would like to draw your attention to a paper by Kok in the *South African Medical Journal* of July last year. He has made some observations which, to my mind, bid fair to put status back in its mythological place. Firstly, he claims to have shown statistically in a large number of normal autopsies that there is no correlation between adrenal weights and thymic weights. In other words, in the absence of gross suprarenal deficiency, people with suprarenal weights in

the lower range of Hamar's and Turnbull's normals do not have thymic weights significantly in excess of those with normal or even high adrenal weights, and *vice versa*. He also describes a large number of experiments with adrenalectomized animals. He found that the adrenalectomized rat is not more liable to sudden death from various forms of trauma while anaesthetized, and that the waking time after ether anaesthesia is not altered by adrenalectomy. Perhaps further experiments along these lines may clarify the position even more. The point that Dr. Griffiths raised about death occurring under anaesthesia in patients after cortisone withdrawal is not, I think, necessarily relevant; they do not die suddenly, as the classical cases of status lymphaticus are alleged to, but go into severe and irreversible hypotension, and die after an interval.

As regards the well known role of *coronory artery* disease in increasing the liability to death on the table, there is little new to add, although perhaps the work of Claude Beck[4] on coronary artery perfusion in dogs may be of interest in this context. Apparently, if one clamps the trachea of an anaesthetized dog with an exposed heart, the whole heart becomes blue, systole becomes irregular and finally the heart stops. If, on the other hand, just one coronary artery is perfused with blue blood while the rest of the heart is receiving red blood, a state of electrical instability arises, and the ventricles are liable to go into fibrillation without warning. As only about 8% of people have any communications between their right and left coronary artery systems, perhaps this might explain the occasional occurrence of sudden death in patients with what appears at autopsy to be relatively mild coronary occlusion, and limited to one artery.

To sum up: When death has come about during operation, and the anaesthetic procedure has contributed to, or caused, that death, there are seldom any pathological changes from which an opinion can be given whether or not the anaesthetic has been properly administered. The value of an autopsy examination in my view is mainly to exclude natural conditions which may have influenced death and to explore the operation site. I do not think it is up to the pathologist to give a cause of death at all unless it is obvious from autopsy findings.

I was very interested to hear that one London Coroner now does not ask the pathologist for any opinion with regard to the anaesthetic procedure, and as well as calling the anaesthetists concerned, when he thinks fit he calls an independent anaesthetist to give an opinion on the clinical history. This seems a very sensible attitude to adopt and could, I think, make the Coroner's inquiry of much greater value to all concerned.

References

1. Nairn, R. C. (1951): J. Path. Bact., **63,** 213.
2. Lucas, B. G. B. (1950): Anaesthesia, **5,** 194.
3. Edwards, G., Morton, H. J. V., Park, E. A. and Wylie, W. D. (1956): Anaesthesia, **11,** 194.
4. Beck, C. (1957): Brit. Med. J., **2,** 1412.

Discussion

Professor Glaister: Before opening the discussion, I would like to make a remark or two regarding the contributions already given. Dr. Platts has thrown light on the academic or research side of the problem and from Dr. Griffiths we have had the fruits of a long experience of the hazards which we are considering this morning. Dr. Mant has flooded us with personal experience and vast numbers of these cases which have been amply supplemented by the contributions of Dr. Hunt. Before calling upon or two of the more senior members present to give short contributions as part of the general discussion I, as the one occupying the Chair would, in the shortest possible terms, like to make comments on three points.

I thought that the reference that Dr. Griffiths made to the pre-operative examination an important one, and over the years that I have had to examine very many anaesthetic deaths it has always been a thought in my mind—the inadequacy of the pre-operative examination. So often, as Dr. Griffiths has said, it falls upon the shoulders of the relatively new members to the medical fold and I have frequently wondered whether now, especially as we have in the institutional aspect, the National Health Service Act, there should not be allocated in surgical wards a more senior member of the staff whose mind clinically is properly attuned to pre-operative examination, particularly in co-operation with the desires of the anaesthetist who has to shoulder the responsibility.

I also want to endorse what Dr. Griffiths has said—I have felt personally my complete inadequacy in having to answer questions following Crown enquiry into anaesthetic deaths. I think there is a class of case where some special enquiries are fruitful in showing some negligence or wrongful medical action but the pathologist is not the proper person to answer such questions that may arise in such a matter and I think in these special cases the forensic pathologists are well advised to ask and insist upon help from specialized colleagues well versed in those specialized spheres. During the last few years I have had some very adverse criticism about medicologists, forensic pathologists, knowing no limit to their knowledge, and I think a lot of these criticisms are well founded.

The third point is: What constitutes, from a purely legal point of view, the death on the table? Does the precise and exact death in your mind satisfy you as an individual? Does that definition satisfy the law? What is the position of a person who, clinically responsible for a case, sees an impending death on the table at the conclusion of an operation and hurriedly gives instructions to get the patient back to bed before he

dies. Is that a death on the table? What happens when the patient is conveyed back to the ward and he dies without regaining consciousness—is that a death on the table? I would like to hear what your views are.

Professor Jepson: It strikes me this morning not only how surprising it is that anyone survives an operation but the great number of causes of death—causes that are foreseeable, predictable or treatable. The subject is so complex and the causes so many, it needs not only a more senior person but a well informed enquiring and careful person to see the patient and to guide him through the operation and post-operative period. This obviously has to be done by the co-operation of the various specialities.

First, the adrenal gland is important in the context of Addison's disease. I have no doubt that a person with Addison's disease tolerates operation extraordinarily badly; but I feel that the adrenal gland is being put a little in the place of the thymus, and that anything that is unexplained is called adrenal failure. As far as I know, nobody has produced data showing that in the patient who dies after operation due to adrenal exhaustion, there is in fact inhibition of secretion of the 17-keto-steroids from the adrenal cortex and I think it might be worth while, in such patients who are so accused of dying, to obtain urine from the bladder or to collect blood to measure the keto-steroids. The only evidence to compare is the fact that some of these patients who go into hypotension do respond to hydrocortisone. Of course, many people with hydrocortisone do respond to noradrenaline, but we do not accuse them of having noradrenaline failure.

The second group of patients who concern me are patients whose heart suddenly stops during the operation—i.e. cardiac arrest. It is said by Stephenson that 300 cardiac arrests occur yearly in this country with fatal consequences. This is a very dramatic occurrence and I think the vast majority of these patients could, in fact, be saved by a quick incision through the chest and massage of the heart. There is one important point about this cardiac massage. I have always been struck by the fact that the heart is a rather soft organ and that one has to be extraordinarily delicate about using the hand to force blood from the left ventricle, and it is very easy to force the finger through the ventricle and to produce bruising or even to rupture the coronary arteries. I am sure, with my own experience, that some of the patients whose hearts have been re-started after cardiac arrest have eventually died from the ventricular trauma inflicted by the surgeon.

Dr. Pleasance: The Association of Anaesthetists have been conducting an enquiry about anaesthetic deaths over the period of 5½ years, trying to find out how they can be prevented. In 1956, out of 1,000 cases investigated, 558 were attributed to the anaesthetic itself. It is a striking thing that of these 558, 110 were produced by the inhalation of vomit —not necessarily solid vomit but liquid, e.g. gastric juice itself. What

interests me is what does form, in certain cases, a sudden death? I do not think we can foretell whether a patient is going to die under the anaesthetic or not. With our cases examined before operation, the routine is that if there is anything abnormal about the patient then a Senior Consultant Anaesthetist usually sees the patient. In the case of a girl with a bad heart, it was suggested to the surgeon that a Senior Physician see the patient. The notes by the physician read: 'I have examined the patient and would advise the administration of gas and oxygen'.

Dr. Camps: I would like to generalize on one or two things. We have had recently a number of deaths in Coloured people. The Coloured population has been increasing and an interesting thing is that in every single one of these cases the patient has expressed a feeling that he will die and also that he does not want an operation. He so often dies in bed afterwards, when you would expect him to be going to survive. This seems to be most unusual. It is a fact with people in West Africa that when the witch doctor says they are going to die, they will die. The same thing happens with regard to pregnancy, and from the morbid or anatomical point of view there is no reason why they should have died. There is nothing in the history, no obstruction, and it is potentially one of the most fascinating investigations.

With regard to the inhalation of stomach contents—an extraordinary state of slow emptying occurs in these pregnant women. I have seen a woman who has been in hospital for 24 hours, who has nothing to eat, and whose stomach was full of undigested rice.

Nobody has mentioned the activities of the surgeon after collapse. A large proportion of these cases are not in the state they were in when they died. I think it is possible in many cases to say what the condition was at the time of collapse. What is a more realistic conception of the cause of death? Vagal inhibition, status thymolymphaticus, these are nothing more or less than an explanation to keep everybody happy. If only one could educate the public and the Coroner that there are many cases where one just does not know the answer and the best thing to do would be to say so. I think it would be most interesting to collaborate with the anaesthetists and see what the anaesthetic investigation could give in evidence. This is one of the cases in which those of you North of the border have a greater advantage than we have in the South. In the North, there is a private enquiry without the publicity. Mackintosh said there is no death on the table that, when the surgeon has been removed from blame, the anaesthetic has not got something to do with. The only answer I can see is that, first of all I do think that in an anaesthetic or operative death the anaesthetist must be present at the autopsy. The anaesthetic forms are most useful but the person must be present himself. The form could be amended. Also there should be the

removal of the anaesthetist being criticized in public. The medico-legal attitude of these anaesthetic deaths must be the desire for proper investigation—an off-the-record discussion, not publicly in court. The pathologist can only go so far. As Professor Jepson has said, how many people die of cardiac massage? Can there not be a joint enquiry—direct communication between this Association and the Faculty of Anaesthetists to give the opportunity of close correspondence and off-the-record discussion?

Professor Glaister: The position in Scotland is not as easy as would appear. Every anaesthetic death has to be reported to the local Procurator and a form of 22 questions filled in, e.g. what was the pre-anaesthetic examination, the resuscitative measures taken, the duration of the anaesthetic, etc. In all these cases the form is sent to the authorities. In a high percentage of cases nothing more is done, but in the odd case instruction will come for a public enquiry.

One case was of a girl suffering from rheumatic endocarditis who had been in the medical ward of a certain hospital. She was taken ill again and arrangements made so that she could be admitted 2 days later to the ward where she had previously been. Early one afternoon 2 days later she was found to be missing. Investigations were made and she was found to have died on the operating table. She had been operated on for an appendicitis and confused with another incoming case. I had the task of investigating it. Had this operation not taken place, might this girl still be alive? What role, following the post-mortem examination, did the anaesthetic play? From the autopsy point of view, do you consider the condition for which she was operated upon an urgent one, in the light of the clinical condition, etc.? I resolved after that case that never again would I ever produce a case before first demanding the presence of a quite unbiased and skilled surgeon and a skilled anaesthetist.

With regard to what Dr. Camps has said about Coloured people dying without apparent cause—I spent many years in Egypt and with regard to medical consultations in the Egyptian government, I had that experience in quite a large number of cases. I am of the opinion that when these Coloured people become sufficiently depressed, they seem to relax all powers of a desire to recover and simply peter out.

Dr. Teare: With regard to Nigerian Coloured people, one should look out for the sequence of labour, anaesthesia, sickle cell anaemia and pulmonary thrombosis. Sickle cell anaemia is sometimes characterized by a spleen the size of a suprarenal and I have seen one, or possibly two such cases.

End of the morning session.

Afternoon Session

An Inquest: Misadventure while Attempting to Take Own Life

Dr. G. J. Griffiths

Mr. President, Gentlemen: On Saturday morning, 23 March last in Derbyshire, the daughter of an old woman, aged 87 years, left her home in company with a medical practitioner for a consultation at Sheffield, leaving the old lady at home by herself. According to the daughter, her mother had suffered from glandular trouble for the past 11 years which took the form of a swelling of the face and this had occurred before breakfast at 8.30 a.m. The swelling had gone when the daughter left home. According to the daughter the mother was a 'happy-go-lucky' woman fond of doing a bit of good to others and never made any suggestion of taking her own life. At 9.30 a.m. the milkman called at the house and saw the old lady putting coal on the fire. He rarely made conversation unless she spoke to him—thus no words were said. He placed the bottles of milk on the kitchen table and went on his way. At 10.10 a.m. the daughter-in-law called and went round to the back of the house and saw the old lady hanging against the wall with her face towards the wall. She had two scarves around her neck and there was a thick cord attached to the scarves. The cord went through the open window and was attached to the banister rail. On the window sill was a pillow. According to her story she sought help from two neighbours, one the doctor's wife and the other a retired matron. The height of the sill from the ground was 10 feet and the height of the old lady was 5 feet 3 inches. Her legs were warm when the daughter-in-law touched them but there was no movement of the body. The daughter-in-law cut the cord while the neighbours held the body. They took the woman indoors and laid her on the settee and called the trainee of the doctor, who arrived at 10.40 a.m. The doctor's wife told the trainee to do nothing until her husband returned from Sheffield. The doctor arrived at about noon, after the wife had telephoned him, and the police were informed about 12.45 p.m. Thus 2 hours had elapsed before the police were informed. The trainee at the inquest was reprimanded for not informing the police when first he saw the body. When the police arrived, the old lady was laid out and devoid of her clothes.

I examined the body of the deceased at the mortuary on Sunday morning 24 March. The body was naked and I was struck by the extreme pallor of the body.

External Appearances: Slight dilatation of the pupils; no cyanosis of the lips, ears or toes; no protrusion or swelling of the tongue; no petechial haemorrhages of the conjunctivae; slight oedema of the legs; superficial markings on the neck on both sides—scratches of the skin, more on the left side. The appearance suggested that the scarves had been behind the lower jaw and the hyoid bone. I was shown the scarves and told by the police that they were not knotted around the neck. The length of the scarves was 1½ feet. No bruising or injury to the side.

Internal Appearances: Apart from arteriosclerotic changes in the circle of Willis, the brain showed nothing; absence of petechial haemorrhages on the epiglottis; no congestion of the trachea. The tissues of the neck showed no evidence of trauma; no fracture of the hyoid bone or cervical vertebrae; no blood-stained fluid in the wind-pipe. There was some terminal congestion of the lungs. The heart was contracted and the 4 chambers devoid of blood. The heart showed myocardial degeneration and the coronary vessels and aorta showed early atheromatous changes.

Examination of the abdomen revealed a large retro-peritoneal haemorrhage and, tracing this back, it was found to have arisen in the origin of the splenic vessels at their junction with the spleen. The spleen itself was ruptured on the anterior surface, but the peritoneal fold was intact. I concluded that death had taken place about 10.05 a.m. on 23 March.

It is known pathologically that during exercise or physical stress, the spleen is capable of dilating and holding almost ½ pint of blood, when the vessels are congested. In view of my finding, I gave the cause of death as retro-peritoneal haemorrhage following traumatic rupture of the spleen. I was asked to give an opinion how this had happened. I told the Coroner that this old lady, in getting out of the window, had sat on the right side of the window-ledge, bringing her legs over the sill and thus compressing the back against the side of the window. When the legs went over the sill, in allowing herself to fall, the sudden weight of the body going over into space, caught the left side of the body against the sharp edge of the sill and thus it received a blow on the spleen, causing it to rupture, the haemorrhage being greater while the organ was congested—she was now hanging with the full weight of the body downwards. The trainee, in his evidence, said that when he examined the body the deceased's corsets had been drawn up over the breast and this, in my opinion, accounted for the lack of injury to the side of the body. I said that in view of the lack of asphyxial changes in the body that the woman was dead from haemorrhage before the full effects from asphyxia by hanging could have taken place.

The Coroner stated that the Jury should have no difficulty in arriving at a decision that this old lady took her own life and she intended to do so by hanging but actually did so by a blow to the spleen which caused the

haemorrhage. The Jury considered the state of her mind and returned a verdict of misadventure while attempting to take her own life. They added there was not sufficient evidence for them to form an opinion about the state of her mind.

In conclusion, an interesting point is this: supposing this old lady was heavily insured and in the policy there was the usual suicide clause—what would the legal consequences be in such a verdict?

Dr. Teare: I am surprised to find a Jury called in this case. It seems to me a great mistake and the Coroner is best able to reach a decision without them. I wonder what the Jury would think of a man who threw himself into the Surrey docks and was rescued by some colleagues. After a short time in a mental hospital he developed jaundice which turned out to be Weil's disease from which he died.

Professional Etiquette

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I hope that in this so-called communication, I am not wasting the time of the members of this Association in bringing to your notice an aspect of professional etiquette to which, in my mind, a changing approach has been made during the last decade. I refer, of course, to the question of professional secrecy in the particular instances where a man may be fit and healthy so far as his workmates and neighbours are concerned and yet, to his doctor's knowledge, a potential menace in his job.

I have thought it right to bring up this subject, largely for discussion, because I have felt that the major portion of the teaching in medical ethics and etiquette in these islands lies with our members and, furthermore, those among us who do not hold teaching appointments are, I know well, frequently called upon for unofficial advice on this very problem. This is a scientific society and, generally speaking, we have avoided making any pronouncement on medical policy. Nevertheless, it could be of considerable help to people like myself who have in their hands the instruction of some 200 undergraduates per annum, not to mention the unlimited potential numbers of postgraduates for advice, to know what our colleagues are thinking and, if possible, to secure some uniformity of attitude towards these problems. There may be diehards among you who will not like what I have to say—others who would go further.

One of the best teaching examples of medical secrecy is contained in the story of the assistant in the country town who, left in charge of his principal's wealthiest patient's wife, performed a vaginal examination and recovered a contraceptive. This I always regard as the epitome of the doctor-patient relationship. The problem of the pregnant housemaid or nanny and the inquisitive mistress or employer is diminishing rapidly. Again the problem of gonorrhoea has largely been extinguished by antibiotics, but how soon may we expect a totally resistant strain, or when may non-specific urethritis be recognized as a venereal disease?

It is perhaps particularly with the epileptic that I am concerned this afternoon, and the epileptic in charge of a vehicle I look on as a real problem. With improved methods of treatment, and increased liberty of the subject, more and more epileptics and mentally unacceptable types are being liberated on the public. What is our attitude to be when seeing an epileptic patient driving the children's bus—warn him or keep silent and wait for the crash?

I hope that what I have had to say will stimulate discussion. I will say quite frankly that I look on the good of the community as being more important than that of the individual.

The position of the British Medical Association has not changed, I think, since 1952 when the following resolution was presented by the Ethical Committee:

'That in general it is a practitioner's obligation to observe strictly the rule of professional secrecy by refraining from disclosing voluntarily without the consent of the patient (save with statutory sanction) to any third party information which he has learnt in his professional relationship with the patient; but that there will doubtless occur certain special occasions when it may become a doctor's moral or social duty for the protection of innocent persons to make disclosure to an interested party if the patient, after having been properly and clearly advised as to the appropriate action which he should take, refuses or fails to do so on his own responsibility'.

This resolution was unfortunately defeated and so far as the B.M.A. is concerned, the old policy of strict professional secrecy is maintained.

So far as disclosure to the police is concerned, the average general practitioner should be on sufficiently good terms with the police to make disclosures in abortion cases if he thinks that there is an abortionist working in the neighbourhood, without mentioning the names of the patients. With regard to other cases I think the advice given by Professor Thompson of Birmingham in the current issue of the *Practitioner* is a good one, where, if ordinary argument with the patient fails, he sends a registered personal letter saying that he can no longer reconcile his duties to the patient with his professional secrecy and asks to be liberated from his professional secrecy. If anyone can advise me as to how I should teach students in this respect, I should be most grateful.

Professor Glaister: I would like to say a little about the difficult problem of a motor driver. I think there is a way out here in the case of epileptics who insist on driving a car to the danger of the public after they have been warned about their condition, and the way out in law, as I see it, is a clear one. We all know that when an individual makes application for a licence to drive a car there is a specific question that demands a specific answer. 'Do you suffer from epilepsy, etc.?' If the applicant deliberately answers in the negative, he is making a false declaration. There is a penalty attached to making a false declaration and it is a simple matter to draw the attention of the authorities to that without any divulgence of professional secrecy.

The question of employees—the doctor should say, 'I have been asked by your employer to examine you—you are under no obligation to have the examination,' but with the patient's permission you divulge what you find. You must have the parent's consent in the case of a minor.

The third group of industrial hazards—the case of hairdressers who have carried on work with a contagious skin disease after having been warned—then the employer should be warned in the matter. Further

examples are: The case of a doctor who sees a patient using a swimming bath after he has been warned not to because of a contagious disease; the signalman who develops signs of insanity, his main complaint being seeing red or green lights during the night. Here he is certifiable. If he had not been certifiable, the guiding principle is that when a set of circumstances exists of such an important character, that justify the practitioner in subordinating his medical duties to his duties as a citizen, then I think that he is covered in a divulgence in these circumstances.

Dr. Imrie: When he comes across an epileptic, the doctor should search his conscience as to whether he reserves his professional secrecy or whether his civic duties necessitate disclosure to the authorities. Take the case of a young fellow driving a bus down a hill with 20 passengers. The boy was 19. He crashed and killed 5 people. He was shown to be an epileptic and found not guilty at the inquest. He was requested to surrender his licence. Where a man like this is a public danger then the doctor could reasonably consider bringing the matter to the attention of the authorities in the hope that in so doing his intentions were in the public welfare and no malice was attached.

Dr. Camps: Can you be sure that an individual will not take action because of the loss of his job? There are two classes. There is the type of case where a person with a disease should not be carrying out his employment. In one case several years ago, a man was investigated in hospital for what appeared to be anginal attacks—quite genuine attacks. His blood pressure at times went up to 400 mm. Hg. He was employed as a long distance express motor coach drive—should he have been employed? This can be applied to some extent to people found to be suffering from infectious diseases, such as bakers, etc.

Professor Glaister: The classical case occurred in Scotland concerning a retired Dr. Pritchard and a Dr. Paterson. Dr. Paterson was called in to treat Pritchard's wife and mother-in-law and, in the course of his evidence in court at the time, explained that, following one of his medical visits, he came to the conclusion that the patient was being practised upon with poison. Considerable cross-examination followed, and he was censored by the Judge that he had failed to disclose what he considered to be a criminal act and he had done wrong. There are many references of sanction where medical witnesses stated that they were not satisfied with the condition of a patient and suspected something. There is a duty on the part of the members of the medical profession to divulge to the proper authorities when they are fully satisfied that a crime has been committed or is being practised, and it becomes a privileged state and should be exonerated. The duties of a general practitioner have been surmounted by the duties of a citizen. It comes as a question of individual interpretation in this case, but the real test must lie with the

doctor. If he feels, as a result of careful thought, that his course of duty as a citizen is to communicate a certain set of circumstances because of its effect on the public, then it is extremely unlikely that any subsequent action will be taken. Even if it is, he will be exonerated by the Jury.

The members of the Board of a large hospital instructed the Superintendent to publish a monthly statement of the staff who were ill. This became a practice, and when I saw the sheet I found the names of the staff, the exact diagnosis of the disease, etc. they were suffering and the period they were under treatment. I said to the Board that it was quite irregular and a breach of professional secrecy, and it was abolished. For the last 2 years the returns still contain the names of the staff, but the diagnosis is left out. Similarly, 2 or 3 weeks ago, the question of a County Council eviction arose. At the last moment it was postponed because the doctor had given a certificate stating that the woman was unfit to be evicted. The County clerk was on the verge of reading out the medical certificate with the condition of the patient. I pointed out what the position was in relation to divulgence and the certificate was not fully read.

Dr. Camps: A small but increasing band of people doing research is sending round circular letters saying they want to investigate a certain condition, e.g. leukaemia. Will members notify the laboratory and fill in the form, etc. This is a possible danger. Research should be done in the office of an organization and not by an individual. Another point raised—the M.O.H. can tell a person to stop work, but he is then responsible for compensation. If you tell an employer and he fails to take action and an accident occurs, then the employer is liable.

Professor Glaister: As a general practitioner knowing that one of your patients is a potential danger, you take no action. Eventually something does happen and there is an enquiry. As a witness you have to divulge and answer questions, and then the question comes, 'Were you aware of this man's condition?' 'Yes.' When did it fall to your knowledge? What action did you take? Did you know he was a public danger? If you warned the patient first, then, if he did not take your advice, you should warn the employer. Then you are in the clear.

Dr. Forbes: With regard to students, tell them something short and snappy or it will not be remembered. An easy approach is to deal with the question of privilege and the privileged actions. Explain the situations which give rise to privileged actions. If the doctor can say to himself, I have a public or private duty which is moral, social or legal, and I am doing this without malice—then that is a qualified privilege. When is an epileptic, an epileptic or not? Can the doctor, on the strength of one fit, go into action? Where do we draw the line?

Dr. Dorward: I have been faced with the same difficulties as Dr. Teare and agree that snappy and short presentations should be made to the students. With regard to case records, it is difficult to advise students. Each practitioner must keep records of his patients and when a patient leaves and transfers to another town, the clinical records must be sent to the new doctor. When a death occurs, the medical records are returned to the authorities as the records are then of no further value to the doctor. The doctors have to enter and sign the date and cause of death on the front of the large medical envelope, and I myself have refused to sign this, as anyone in the Births and Deaths Registration Office can read this envelope.

Professor Glaister: Whenever any particular procedure becomes law, then all sections and sub-sections within that Act become the controlling features of citizenship, and whenever there is a controlling Act laying down procedure you will be exempt from any actions arising from it (when it gives exoneration).

The issuing of certificates occurs in the Births and Deaths Registration Act. It is the duty of certain people to issue these certificates. It is laid down within the Act where the certificate goes and after that the individual discharges his responsibility. If this particular procedure you mention is specifically mentioned with the National Health Service Act, you are covered in carrying out the stipulations of that Act.

A Member: With regard to leukaemia: in my experience physicians do not very often tell their patients that they have leukaemia. In relation to the nursing staff: in some cases, e.g. a simple matter of blood counts, these pass through various hands with people reading them—but they may be Wassermann reactions and therefore the reports should be put in a sealed envelope and only opened by a person in charge.

Dr. Marshall: In those cases where the decision has to be made with employers or authorities, we teach students first to ask their Defence Society for advice.

Professor Glaister: How often do you get negative results?

Dr. Evans: Do you include *petit mal*?

Dr. Griffiths: Consider peripheral neuritis. When a surgeon is called to examine a patient whom he knows to be a chronic alcoholic, what is his position with divulging peripheral neuritis?

Dr. Teare: I am delighted to hear that Dr. Marshall advised the students to get in touch with the Medical Defence Society. I believe that the safety of the public is more important than the private individual. I would expose *petit mal* and epilepsy, providing I could do so with a clear conscience.

Professor Glaister: Every case, as it arises, must receive consideration, but that consideration must be based on the ascertained law.

Is Murder Possible in England Under the Cremation Act?

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Since the new Cremation Act, there has been some anxiety felt about the value of the C Certificate. It is suggested that on occasions it is unsatisfactory because of the manner in which it is completed, but I understand from the authorities that this is no reason for discarding it. I therefore investigated how exactly it is filled in. The doctor who is qualified to sign it has to answer a number of questions including have you examined the body? and have you talked to certain people *alone?* He must moreover not be in a certain relationship with the person who has signed the B Certificate.

These certificates were framed many years ago and it is of interest to consider how satisfactory they are under present-day conditions. I think they are first class if correctly done. Under the National Health Act, a person under whole-time contract with the NHS has to fill in the B Certificate free but the C Certificate comes outside the terms of service and commands a fee. Further, a curious anomaly exists in hospitals whereby a senior member of the staff may complete the C Certificate in a case in which the B Certificate has been completed by one of his juniors. It is difficult to see, in such circumstances, why it is laid down that the person who completes the C Certificate shall not be in partnership with the signatory of the B Certificate.

The main criticism, however, has been as to its accuracy.

An unofficial census of doctors shows that a proportion of C Certificates is filled in without the body having been looked at, at all. Others are completed without the body having been examined. Here the certificate is failing in its function. There is already a loop-hole in death certificates, for no doctor is compelled to see the body after death. If he is then prepared to sign the certificate, there is no proof of identity. A complaint of conscientious doctors asked to sign certificate C is that they have been willing to examine the body but have been unable to do so as it has already been screwed down in the coffin. No undertaker has actually refused to unscrew it—but some have appeared very reluctant and said that it was the first time doctors had ever asked them to do so. Although the external examination of the body by the man who signs the C Certificate is in many

cases valueless, nevertheless the presence of hypodermic injection marks may lead to suspicion. There is, therefore, good reason for the external examination. There was a case in the U.S.A. of a body shipped from one state to another with several bullet holes; whilst even here a body was taken to the public mortuary with a stocking still tied around its neck, which the doctor apparently had not noticed.

Some doctors appear to be obtaining money under false pretences, but often these certificates are not a paying proposition for an ordinary general practitioner, for they involve visits and appointments. That is no reason for it to be generally accepted that the C Certificate is only a testimonial to the man who signed the B Certificate.

What alternative is there? Money paid out for C Certificates may be somewhere around £3,000,000 per year. The payment to the referee in England is nothing compared to that paid for C Certificates and it is the referee who should pick out the flaws. It is of interest that the referee is now commonly the M.O.H. In some districts he gets a fee per case—in others it is covered by his salary. In a certain city in England there are 4,000 cremations a year and the M.O.H. is the referee—it might be asked, when does he find time to do his work as M.O.H.?

There is variation in the standard of referees and hence it is theoretically possible for a doctor who does not want a certificate questioned to send it to a referee who, he knows, is most tolerant. Thus cases recorded as having died with gastro-enteritis have been passed without any hesitation. It could be easy to dispose of the wrong body. There is, moreover, no law to prevent you, if the cremation certificate is issued, from whipping the body away and burying it. Embalming also does not help.

Unless it is insisted that the referees have some experience medico-legally, it would seem that some might not recognize a case of poisoning or homicide and unless they have some suspicions, it might be easy to deceive them. Thus there is ample scope for misuse.

In Denmark cremation is carried out entirely under a doctor's death certificate and when the matter is scrutinized it is found that the standard required on death certificates is considerably higher than is required in England at the moment.

As an example, the following case is quoted: 'A lady some years ago committed suicide by gassing herself. The death was notified to the Coroner who discovered she had died 2 days after her husband. Her husband proved to be alleged only; but a cremation certificate had been issued by the 2 doctors giving the cause of death as cerebral haemorrhage due to arterio-sclerosis and hypertension. The Coroner, feeling there might be an association between the two deaths, stopped the cremation and had an autopsy carried out. On external examination the deceased was deeply jaundiced and had such an enormous mass of secondaries in his liver that it could not possibly have been missed if examined. He had died of carcinomatosis.

She had run away 20 years before to live with him. A will had been made in her favour. Her true husband was at first reluctant to identify the body but the outcome was that he inherited £20,000 as a result of her death.'

Professor Glaister. Despite a certain urge on the part of the government to abolish the 'C' certificate, the authorities were dead against it and with them I sided. Dr. Camps has painted a dramatic picture and it is not for me to say how correct or incorrect that may be as applicable to English procedure. As a referee I would never authorize a cremation of any body that had been embalmed, before the full procedure had been carried out.

Certificate B: Is there any evidence of gastro-enteritis? If that question is not answered, the cremation is held up on my authority until it is answered in the negative, following personal enquiry by me. If an enquiry is held we have an understanding with the Procurator Fiscal, and co-operation of the staff, etc. If we refer a case to them, they take it up and make their enquiries and only after they are satisfied do they issue a clearance certificate form E.1. When invesigating the case, only when you are satisfied, should a certificate be given and only when every question is answered. I have only once over the years in Scotland encountered one case which was to my knowledge, presented in association with faked documents.

I have come across cases in which Certificate 'C' has been completed defectively. I will give you two examples. I happened to be telephoning a woman doctor who had given a certificate and she was well over the 5 years' qualification necessary. I told her I was telephoning to ask her a few questions about the certificate. During the course of the conversation she told me that she had had pleurisy for the last 5 weeks, and was still not feeling too well. I asked: How did you manage to give a 'C' certificate? After a few moments she broke down—she had given the certificate blindly on what the relatives had told her. I said, the cremation certificate will be extracted from the file of papers and retained by me. We got the assistant who had seen the case, and the certificate was substituted.

I close on this note. A doctor in Aberdeen a few years ago was charged in the criminal court with having issued 5 confirmatory certificates. The charge was that he had not interviewed the doctor who had issued the first certificate.

Dr. Camps: The problem still remains whether this C certificate could be done away with.